

Treatment of Extra-Abdominal Desmoid Tumors With Interferon-Alpha With or Without Tretinoin

ANDREAS LEITHNER, MD,¹ BEATE SCHNACK, MD,² THOMAS KATTERSCHAFKA, MD,¹
CHRISTOPH WILTSCHKE, MD,² GABRIELE AMANN, MD,³ REINHARD WINDHAGER, MD,⁴
RAINER KOTZ, MD,¹ AND CHRISTOPH C. ZIELINSKI, MD^{2,5,6*}

¹Department of Orthopedic Surgery, University Hospital, Vienna, Austria

²Clinical Division of Oncology, Department of Medicine I, University Hospital, Vienna, Austria

³Clinical Division of Clinical Pathology, Department of Pathology, University Hospital, Vienna, Austria

⁴Department of Orthopedic Surgery, Karl Franzens University, Graz, Austria

⁵Division of Medical Experimental Oncology, Department of Medicine I, University Hospital, Vienna, Austria

⁶Ludwig Boltzmann Institute for Clinical Experimental Oncology, Vienna, Austria

Background and Objectives: Surgery is the main treatment for extra-abdominal desmoid tumors, but the results of further management remain uncertain. Therefore, a retrospective analysis was undertaken to evaluate the toxicity and efficacy of treatment with interferon-alpha (IFN- α) \pm tretinoin in this setting.

Methods: Thirteen patients with extra-abdominal desmoid tumors and a median age of 32 years (range, 15–73) received IFN- α . Seven of these patients received a combination of IFN- α and tretinoin in order to test further enhancement.

Results: After a mean observation period of 27 ± 15 months (mean \pm standard deviation) under treatment with IFN- α \pm tretinoin, local control was seen in 11 of 13 patients (85%). Seven patients had no evidence of disease at a mean disease-free interval of 22 ± 18 months; in two patients progressive disease occurred after only 7 and 9 months, respectively, of observation. In another four patients, progression of the desmoid tumor was stabilized.

Conclusions: The data of this retrospective, nonrandomized study on therapy with IFN- α \pm tretinoin suggest that such treatment may be effective in prolonging the disease-free interval of patients after intralesional or marginal surgery. Because of the encouraging response rate, this regimen appears to be another nonsurgical treatment alternative.

J. Surg. Oncol. 2000;73:21–25. © 2000 Wiley-Liss, Inc.

KEY WORDS: desmoids; aggressive fibromatosis; systemic therapy; nonsurgical treatment

INTRODUCTION

Desmoid tumors—also known as aggressive fibromatoses—are rare (3–4 per million per year) [1], benign, but locally aggressive soft-tissue neoplasms of unknown etiology [2]. An extra-abdominal form is distinguished from an intra-abdominal one. Although they do not metastasize or dedifferentiate, local invasion and destruction

constitute frequent problems. Desmoid tumors occur in nearly every part of the body and often recur after sur-

*Correspondence to: Christoph C. Zielinski, MD, Chair for Medical Experimental Oncology, Department of Medicine I, University Hospital, 18-20 Währinger Gürtel, A-1090 Vienna, Austria. Fax: **43/1/40400-4452. E-mail: Christoph.Zielinski@akh-wien.ac.at

Accepted 24 September 1999

TABLE I. Characteristics of Patients With Desmoid Tumors Treated With IFN- α \pm Tretinoin

No.	Age at first diagnosis (years)	Gender	Site	Margins at last surgery	Type of tumor	No. of previous recurrences
1	36	F	Buttock	Intralesional	Recurrent	3
2	44	M	Leg	Marginal	Recurrent	3 (last multiple)
3	45	F	Arm	Wide	Recurrent	1
4	21	F	Chest	Marginal	Recurrent	1
5	25	M	Buttock	Intralesional	Primary	
6	30	M	Leg	Marginal	Recurrent	2
7	57	F	Chest	Wide	Primary	
8	19	F	Arm	Marginal	Primary	
9	9	F	Buttock	Marginal	Recurrent	6
10	32	F	Leg	Intralesional	Recurrent	1
11	31	F	Leg	Intralesional	Recurrent	2
12	14	M	Arm	Intralesional	Recurrent	1
13	26	F	Leg	Biopsy only	Primary	

gery, particularly after marginal or intralesional excision. Because of these characteristics, desmoid tumors are sometimes also referred to as low-grade fibrosarcomas [3,4].

While optimal management is largely uncertain, patients with desmoid tumors have been treated with various combinations of surgery, radiotherapy, cytotoxic chemotherapy, nonsteroidal anti-inflammatory drugs, and anti-estrogens. Due to the side effects and/or unsatisfying response rates of most measures, other nonsurgical alternatives are being investigated.

Based on a case report of a positive effect of interferon-alpha (IFN- α) on an abdominal desmoid tumor [5] and on in vitro data on the antiproliferative effects of retinoic acid and IFN- α on fibroblasts [6,7], it seemed appropriate to use IFN- α in patients with desmoid tumors after intralesional or marginal surgery, after recurrence, or in lesions of considerable extent. Thus, a retrospective analysis and literature review were undertaken to evaluate the possibility of improving the surgical treatment of desmoid tumors by using IFN- α \pm tretinoin.

MATERIALS AND METHODS

Patients and Previous Treatment

Thirteen patients (4 males, 9 females) with histologically confirmed extra-abdominal desmoid tumors were included in the study after obtaining informed consent. Gardner's syndrome was not observed in this series. The median age at first diagnosis was 30 years (range, 9–57), at the start of treatment with IFN- α it was 32 years (range, 15–73). There were 5 patients with tumors in the leg, 3 with tumors in the buttocks, 2 with tumors in the chest, and 3 with tumors in the arm (Table I).

Of the 13 patients, 9 had undergone one to six resections due to progressive disease previously. One patient (no. 11) had received radiotherapy in 1955 due to the erroneous assumption of the presence of fibrosarcoma.

None of the remaining 8 patients had ever received radiotherapy or cytotoxic chemotherapy.

Observation and Follow-Up

Clinical follow-up consisted of laboratory analysis including peripheral blood cell counts (leukocytes, thrombocytes, and erythrocytes) and the assessment of liver and kidney function parameters combined with physical examination in 1- to 2-month intervals. In order to assess tumor volume, periodic computed tomography or magnetic resonance imaging was performed in intervals of 3–6 months or when there was clinical suspicion of progressive disease (PD). Any increase of >25% in the size of the desmoid tumor or signs of recurrent disease was considered a treatment failure in the sense of PD.

Treatment

All 13 patients received 15 μ g recombinant IFN- α -2c (Berofor®, Boehringer Ingelheim, Bender, Vienna) corresponding to 3,500,000 IU (World Health Organization standard for recombinant IFN- α -2c) three times weekly by subcutaneous injection on a continuing schedule. The mean treatment period was 16 ± 8 months (mean \pm standard deviation). Seven patients received a combination of IFN- α and tretinoin in order to test further enhancement of the efficacy of adjuvant treatment with IFN- α . The daily oral dosage of tretinoin was 30 mg administered in parallel to IFN- α on a continuing basis.

As shown in Table II, 9 patients received IFN- α \pm tretinoin as an adjuvant treatment in conjunction with surgery, 3 after first surgery and 6 after surgery of a recurrent lesion. In all 9 patients, the risk of recurrence was judged to be extremely high based on either previous recurrences and/or an intralesional or marginal excision at last surgery. Margins at last surgery were intralesional in 2, marginal in 5, and wide in 2. Four

TABLE II. Results of Treatment of Desmoid Tumor Patients Receiving IFN- α \pm Tretinoin

No.	Age at start of treatment with IFN- α (years)	Duration of Treatment with IFN- α (mos.)	Aim of treatment with IFN- α	Additional therapy	Response	Progression after (mos.)	DFI (mos.)	Follow-up from start of treatment with IFN- α (mos.)
1	41	19	Prevent recurrence		NED		68	58
2	52	15	Prevent recurrence	Tretinoin	NED		37	36
3	46	12	Prevent recurrence	Tretinoin	NED		15	15
4	22	20	Prevent recurrence	Tretinoin	NED		20	20
5	25	9	Prevent recurrence		PD	9	9	31
6	31	11	Prevent recurrence	Tretinoin	NED		11	11
7	57	19	Prevent recurrence		NED		19	19
8	19	7	Prevent recurrence		PD	7	7	7
9	18	10	Prevent recurrence		NED		12	10
10	32	36	Stabilize PD	Tretinoin	SD			36
11	73	26	Stabilize PD	Tretinoin	SD			37
12	15	9	Stabilize PD	Tretinoin	SD			31
13	36	12	Stabilize PD		SD			35

patients received a combination of IFN- α and five were treated with IFN- α alone.

Four patients received IFN- α \pm tretinoin to stabilize PD. Three patients received a combination of IFN- α and tretinoin and one was treated with IFN- α alone. One patient (no. 13) had a biopsy performed and refused further surgical treatment. Nineteen months prior to the start of treatment with IFN- α , he was treated unsuccessfully with colchicine for 3 months. Progression of the size of the lesion was confirmed by magnetic resonance imaging. None of the remaining 3 patients had ever received radiotherapy or cytotoxic chemotherapy.

RESULTS

After a mean observation period of 27 ± 15 months, local control was observed in 11 of 13 patients (85%) treated with IFN- α \pm tretinoin. All patients were alive at the time of analysis and no patient was lost to follow-up.

Adjuvant Treatment to Prevent Recurrence

Nine patients received adjuvant treatment following surgery. Seven patients had no evidence of disease (NED) at a mean disease-free interval (DFI) of 22 ± 18 months. PD occurred in the 2 remaining patients after only 7 and 9 months, respectively, of observation. Thus, the mean DFI under adjuvant treatment with IFN- α \pm tretinoin was 22 ± 18 months.

Treatment With IFN- α to Stabilize PD

Four patients were treated with IFN- α \pm tretinoin to stabilize PD. At a mean observation period of 35 ± 3 months, progression of the desmoid tumor was stabilized in all four patients.

Treatment-Associated Toxicity

Side effects of treatment with IFN- α alone or in conjunction with tretinoin were infrequent and generally mild. In patient 11, sicca syndrome was diagnosed after

12 months of treatment with IFN- α and tretinoin. As this complication was ascribed to tretinoin, the patient was continued on IFN- α alone. In patient 9, it was necessary to interrupt therapy with IFN- α due to severe nausea and fever. No other therapy was given.

DISCUSSION

Optimal management of desmoid tumors (aggressive fibromatosis) is still controversial [8–11] and local control is often extremely difficult to achieve [12]. Surgery is the mainstay of therapy. However, recurrence rates ranging from 31% to 68% have been reported [3,9,12–19], with recurrence leading to more aggressive surgical procedures or even amputations of limbs [9,14,15]. Most studies prove that margin status is the most important predictor of local recurrence for patients with resectable, unifocal desmoid tumors [2,8,18,20–22]. About 90% of local recurrences occur within the first 2 years [9,15–17].

In 1984, Rock et al. [15] found that the recurrence rate was approximately 90% in a series of 194 patients who had local marginal or intralesional excisions. Most of the patients in our study were patients with recurrent tumors and intralesional or marginal resections. Despite these poor prognostic factors, our results suggest that therapy with IFN- α \pm tretinoin may provide improvement in local control.

However, the small number of patients is a limitation of this retrospective analysis. Desmoid tumors are extremely rare—an incidence of three to four per 10^6 or 1,000,000 inhabitants per year has been reported for the Finnish population [1]. In most cases, the rarity of this tumor has prohibited the accumulation of a large series. Mainly case reports [23–43] and small series (<42 patients) [3,5,8,9,14,19,22,44–57] have been reported in the literature. Larger series (45–194 patients) only appeared in conjunction with radiotherapy and/or surgery [4,12–14,17,20,21,58,59].

Therefore, the comparison of the present therapy with

other treatment modalities is difficult. Radiotherapy has been successfully used, either alone in patients with unresectable or inoperable disease [4,18,47,54,58–60] or following surgery [4,5,8,18,20,21,22,40,47,53,54,58–60]. Interstitial brachytherapy has been used as well [50,52,61]. Other authors, however, have judged radiation to be of limited value [14,15,19,29,46,48,51].

Similarly, chemotherapy including doxorubicin, vinblastine, methotrexate, and dacarbazine has been associated with positive response rates [27,28,31,32,34,37,42,44,56]. Other series have claimed no benefit with cytotoxic therapy [33,51]. Furthermore, in conjunction with radiotherapy and cytotoxic chemotherapy, severe side effects like wound healing problems and other potentially adverse effects like the risk of second malignancy have to be kept in mind [4,5,15,16,22,27,31,33,51,52,61,62].

Finally, some authors have also reported objective responses following endocrine manipulations with antiestrogen [23,25,30,36,38,41,43,55,63], antiprostaglandin [55], and nonsteroidal anti-inflammatory drugs [24,26,45,49,55]. Other authors, however, reported treatment failures [5,22,26,39,63].

Due to the side effects and/or unsatisfying response rates of most measures, other nonsurgical alternatives were attempted. As periods of stable disease (SD) without progression and few spontaneous regressions have been reported [13,16,17,22,35,53,64], a nonaggressive treatment was needed to achieve longer DFI or SD.

The regression of a great abdominal desmoid tumor by IFN- α [39] and long-term regression of a recurrent mesenteric desmoid tumor after IFN- γ therapy have been reported [11]. Furthermore, in vitro investigations and animal experimental data suggest an antiproliferative effect of both IFN- α and retinoic acid on fibroblasts [6,7]. Therefore, the 13 patients included in our study were treated with IFN- α alone or with a combination of IFN- α and tretinoin.

The policy at our institutions is to employ surgery as the primary treatment modality. Following surgery, we recommend a nonaggressive management approach using IFN- α \pm tretinoin for 12 months. Radiotherapy and/or cytotoxic drug therapy should be reserved for the management of progressive or inoperable disease. As for the teratogenic potential of tretinoin, pregnancy has to be avoided or, in case of uncertainty, this drug must not be used.

In conclusion, the data of this retrospective, nonrandomized study on the treatment of extra-abdominal desmoid tumors with IFN- α \pm tretinoin suggest that such therapy may be effective in prolonging the DFI of patients after intralesional or marginal surgery. Moreover, in 4 patients, progressive desmoid tumors could be stabilized for considerable periods by the use of IFN- α \pm tretinoin. Because of the encouraging response rates in

11 of 13 patients (85%), this regimen appears to be another nonsurgical treatment alternative.

REFERENCES

1. Reitamo JJ, Hayry P, Nykyri E, et al.: The desmoid tumor. I. Incidence, sex, age and anatomical distribution in the Finnish population. *Am J Clin Pathol* 1982;77:665–673.
2. Fisher C: Fibromatosis and fibrosarcoma in infancy and childhood. *Eur J Cancer* 1996;32A:2094–2100.
3. Lopez R, Kemalyan N, Moseley HS, et al.: Problems in diagnosis and management of desmoid tumors. *Am J Surg* 1990;159:450–453.
4. Sherman NE, Romsdahl M, Evans H, et al.: Desmoid tumors: A 20-year radiotherapy experience. *Int J Radiat Oncol Biol Phys* 1990;19:37–40.
5. Acker JC, Bossen EH, Halperin EC: The management of desmoid tumors. *Int J Radiat Oncol Biol Phys* 1993;26:851–858.
6. Gabbert HE, Gerharz CD, Biesalski HK, et al.: Terminal differentiation and growth inhibition of a rat rhabdomyosarcoma cell line (BA-HAN-1C) in vitro after exposure to retinoic acid. *Cancer Res* 1988;48:5264–5269.
7. Balkwill FR, Bokhonko AI: Differential effects of pure human alpha and gamma interferons on fibroblast cell growth and the cell cycle. *Exp Cell Res* 1984;55:190–197.
8. Karakousis CP, Mayordomo J, Zografos GC, et al.: Desmoid tumors of the trunk and extremity. *Cancer* 1993;72:1637–1641.
9. Higaki S, Tateishi A, Ohno T, et al.: Surgical treatment of extra-abdominal desmoid tumours (aggressive fibromatoses). *Int Orthop* 1995;19:383–389.
10. Suit HD: Radiation dose and response of desmoid tumors. *Int J Radiat Oncol Biol Phys* 1990;19:225–227.
11. Bauernhofer T, Stoger H, Schmid M, et al.: Sequential treatment of recurrent mesenteric desmoid tumor. *Cancer* 1996;77:1061–1065.
12. Pritchard DJ, Nascimento AG, Petersen IA: Local control of extra-abdominal desmoid tumors. *J Bone Joint Surg [Am]* 1996;78:848–854.
13. Reitamo JJ: The desmoid tumor. IV. Choice of treatment, results, and complications. *Arch Surg* 1983;118:1318–1322.
14. Enzinger FM, Shiraki M: Musculo-aponeurotic fibromatosis of the shoulder girdle (extra-abdominal desmoid). Analysis of thirty cases followed up for ten or more years. *Cancer* 1967;20:1131–1140.
15. Rock MG, Pritchard DJ, Reiman HM, et al.: Extra-abdominal desmoid tumors. *J Bone Joint Surg [Am]* 1984;66:1369–1374.
16. Miralbell R, Suit HD, Mankin HJ, et al.: Fibromatoses: From postsurgical surveillance to combined surgery and radiation therapy. *Int J Radiat Oncol Biol Phys* 1990;18:535–540.
17. Markhede G, Lundgren L, Bjurstam N, et al.: Extra-abdominal desmoid tumors. *Acta Orthop Scand* 1986;57:1–7.
18. Spear MA, Jennings LC, Mankin HJ, et al.: Individualizing management of aggressive fibromatoses. *Int J Radiat Oncol Biol Phys* 1998;40:637–645.
19. Plukker JT, Oort IV, Vermey A, et al.: Aggressive fibromatosis (non-familial desmoid tumour): Therapeutic problems and the role of adjuvant radiotherapy. *Br J Surg* 1995;82:510–514.
20. Goy BW, Lee SP, Eilber F, et al.: The role of adjuvant radiotherapy in the treatment of resectable desmoid tumors. *Int J Radiat Oncol Biol Phys* 1997;39:659–665.
21. Ballo MT, Zagars GK, Pollack A: Radiation therapy in the management of desmoid tumors. *Int J Radiat Oncol Biol Phys* 1998;42:1007–1014.
22. Mitchell G, Thomas JM, Harmer CL: Aggressive fibromatosis: Evidence for a stable phase. *Sarcoma* 1998;2:149–154.
23. Brooks MD, Ebbs SR, Colletta AA, Baum M: Desmoid tumours treated with triphenylethylenes. *Eur J Cancer* 1992;28A:1014–1018.
24. Waddell WR, Gerner RE: Indomethacin and ascorbate inhibit desmoid tumors. *J Surg Oncol* 1980;15:85–90.
25. Kinzbrunner B, Ritter S, Domingo J, et al.: Remission of rapidly

- growing desmoid tumors after tamoxifen therapy. *Cancer* 1983;52:2201–2204.
26. Klein WA, Miller HH, Anderson M, et al.: The use of indomethacin, sulindac, and tamoxifen for the treatment of desmoid tumors associated with familial polyposis. *Cancer* 1987;60:2863–2868.
 27. Klaase JM, Kroon BB, Benckhuijsen C, et al.: Results of regional isolation perfusion with cytostatics in patients with soft tissue tumors of the extremities. *Cancer* 1989; 64:616–621.
 28. Weiss AJ, Lackman RD: Low-dose chemotherapy of desmoid tumors. *Cancer* 1989;64:1192–1194.
 29. Atahan IL, Akyol F, Zorlu F, et al.: Radiotherapy in the management of aggressive fibromatosis. *Br J Radiol* 1989;62:854–856.
 30. Sportiello DJ, Hoogerland DL: A recurrent pelvic desmoid tumor successfully treated with tamoxifen. *Cancer* 1991;67:1443–1446.
 31. Reich S, Overberg-Schmidt US, Buhner C, et al.: Low-dose chemotherapy with vinblastine and methotrexate in childhood desmoid tumors. *J Clin Oncol* 1999;17:1086.
 32. Hamilton L, Blackstein M, Berk T, et al.: Chemotherapy for desmoid tumours in association with familial adenomatous polyposis: A report of three cases. *Can J Surg* 1996;39:247–252.
 33. Schnitzler M, Cohen Z, Blackstein M, et al.: Chemotherapy for desmoid tumors in association with familial adenomatous polyposis. *Dis Colon Rectum* 1997;40:798–801.
 34. Lynch HT, Fitzgibbons R, Chong S, et al.: Use of doxorubicin and dacarbazine for the management of unresectable intra-abdominal desmoid tumors in Gardner's syndrome. *Dis Colon Rectum* 1994; 37:260–267.
 35. Maurer F, Horst F, Pfannenberger C, et al.: Multifocal extra-abdominal desmoid tumor: Diagnostic and therapeutic problems. *Arch Orthop Trauma Surg* 1996;115:359–362.
 36. Procter H, Singh L, Baum M, et al.: Response of multicentric desmoid tumours to tamoxifen. *Br J Surg* 1987;74:401.
 37. Douglass HO Jr, Karakousis C: Alternating administration of Adriamycin (NSC-123127) and vincristine (NSC-67574)-actinomycin D (NSC-3053) in advanced sarcomas. *Cancer Chemother Rep* 1975;59:1045–1047.
 38. Maroy B: Tumeur desmoïde sensible au tamoxifène. *Presse Med* 1997;26:1520–1522.
 39. Geurs F, Kok TC: Regression of a great abdominal desmoid tumor by interferon alpha. *J Clin Gastroenterol* 1993;16:264–265.
 40. Hill DR, Newman H, Phillips TL: Radiation therapy of desmoid tumors. *Am J Roentgenol Radium Ther Nucl Med* 1973;117:84–89.
 41. Lackner H, Urban C, Kerbl R, et al.: Noncytotoxic drug therapy in children with unresectable desmoid tumors. *Cancer* 1997;80:334–340.
 42. Seiter K, Kemeny N: Successful treatment of a desmoid tumor with doxorubicin. *Cancer* 1993;71:2242–2244.
 43. Thomas S, Datta GS, Kapur BM: Treatment of recurrent desmoid tumour with tamoxifen. *Aust N Z J Surg* 1990;60:919–921.
 44. Patel SR, Evans HL, Benjamin RS: Combination chemotherapy in adult desmoid tumors. *Cancer* 1993;72:3244–3247.
 45. Waddell WR, Gerner RE, Reich MP: Nonsteroid antiinflammatory drugs and tamoxifen for desmoid tumors and carcinoma of the stomach. *J Surg Oncol* 1983;22:197–211.
 46. Kiel KD, Suit HD: Radiation therapy in the treatment of aggressive fibromatoses (desmoid tumors). *Cancer* 1984;54:2051–2055.
 47. Keus R, Bartelink H: The role of radiotherapy in the treatment of desmoid tumours. *Radiother Oncol* 1986;7:1–5.
 48. McKinnon JG, Neifeld JP, Kay S, et al.: Management of desmoid tumors. *Surg Gynecol Obstet* 1989;169:104–106.
 49. Waddell WR, Kirsch WM: Testolactone, sulindac, warfarin, and vitamin K1 for unresectable desmoid tumors. *Am J Surg* 1991; 161:416–421.
 50. Schmitt G, Mills EE, Levin V, et al.: Radiotherapy of aggressive fibromatosis. *Eur J Cancer* 1992;28A:832–835.
 51. Tsukada K, Church JM, Jagelman DG, et al.: Systemic cytotoxic chemotherapy and radiation therapy for desmoid in familial adenomatous polyposis. *Dis Colon Rectum* 1991;34:1090–1092.
 52. Zelefsky MJ, Harrison LB, Shiu MH, et al.: Combined surgical resection and iridium 192 implantation for locally advanced and recurrent desmoid tumors. *Cancer* 1991;67:380–384.
 53. Goy BW, Lee SP, Fu YS, et al.: Treatment results of unresected or partially resected desmoid tumors. *Am J Clin Oncol* 1998;21: 584–590.
 54. McCollough WM, Parsons JT, van der Griend R, et al.: Radiation therapy for aggressive fibromatosis. The Experience at the University of Florida. *J Bone Joint Surg [Am]* 1991;73:717–725.
 55. Tsukada K, Church JM, Jagelman DG, et al.: Noncytotoxic drug therapy for intra-abdominal desmoid tumor in patients with familial adenomatous polyposis. *Dis Colon Rectum* 1992;35:29–33.
 56. Weiss AJ, Horowitz S, Lackman RD: Therapy of desmoid tumors and fibromatosis using vinorelbine. *Am J Clin Oncol* 1999;22: 193–195.
 57. Cole NM, Guiss LW: Extra-abdominal desmoid tumors. *Arch Surg* 1969;98:530–533.
 58. Stockdale AD, Cassoni AM, Coe MA, et al.: Radiotherapy and conservative surgery in the management of musculo-aponeurotic fibromatosis. *Int J Radiat Oncol Biol Phys* 1988;15:851–857.
 59. Kamath SS, Parsons JT, Marcus RB Jr, et al.: Radiotherapy for local control of aggressive fibromatosis. *Int J Radiat Oncol Biol Phys* 1996;36:325–328.
 60. Catton CN, O'Sullivan B, Bell R, et al.: Aggressive fibromatosis: Optimisation of local management with a retrospective failure analysis. *Radiother Oncol* 1995;34:17–22.
 61. Assad WA, Nori D, Hilaris BS, et al.: Role of brachytherapy in the management of desmoid tumors. *Int J Radiat Oncol Biol Phys* 1986;12:901–906.
 62. Raney RB Jr: Chemotherapy for children with aggressive fibromatosis and Langerhans' cell histiocytosis. *Clin Orthop* 1991;262: 58–63.
 63. Wilcken N, Tattersall MH: Endocrine therapy for desmoid tumors. *Cancer* 1991;68:1384–1388.
 64. Jenkins NH, Freedman LS, McKibbin B: Spontaneous regression of a desmoid tumour. *J Bone Joint Surg [Br]* 1986;68:780–781.